A PROGRAM FOR THE DISPLAY OF THREE DIMENSIONAL ELECTROPHYSIOLOGIC DATA

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A major limitation in the elucidation of the mechanisms of ventricular fibrillation and defibrillation is the lack of adequate means of viewing the results of experimental and clinical studies in a realistic and understandable manner. The data to be displayed are inherently three dimensional and are ofter acquired from widely spaced electrodes, requiring interpolation and three dimensional computer graphics techniques. We have developed a set of programs to display potentials and the magnitudes of the estimated potential gradients generated by defibrillation shocks as well as activation sequences in a variety of experimental conditions. There are three types of displays. The first type displays three-dimensional surfaces constructed from triangularized electrode locations. Potentials or gradient magnitudes are interpolated over each surface and are displayed as spatial changes in intensity or hue. The second type of display is a volume reconstruction of the myocardium. In this display, spatial changes in potentials or gradient magnitudes in the myocardial volume are represented by spatially varying the hues of the volume elements. The values of most volume elements are interpolated with a three-dimensional method based on discrete smooth interpolation. The third type of display is a three-dimensional surface reconstruction of the heart on which are mapped the interpolated values of the epicardial and endocardial boundaries of the volume representation. The values are represented by variations in hue. The visual effects are enhanced by interactive manipulation of the displays on a high speed graphics processor, allowing viewing from a variety of angles and in different planes through the myocardium. The displays have proved to be extremely helpful in visualizing the electrophysiological phenomena that are associated with cardiac arrhythmias, and with the study of fibrillation and defibrillation.

A COMPUTER-AIDED APPROACH FOR THE SPATIAL AND TEMPORAL QUANTITATION OF LEFT VENTRICULAR ENDOCARDIAL CONTOUR AND MOTION USING TWO-DIMENSIONAL ECHOCARDIOGRAPHY.

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Quantitation of regional function is becoming increasingly important for assessing the effects of interventions in acute ischemic syndromes. The inclusion of the entire systolic contraction sequence has been demonstrated to be superior to analysis of just end-diastolic and end-systolic frames for this purpose. Manual tracing of the endocardial contours, however, is time-consuming, tedious, and observer dependent. To overcome these limitations, we have developed an approach where the observer places only 10-15 points along the endocardial circumference (depending upon the quality of the two-dimensional echocardiographic images) and the contour is automatically derived by natural spline interpolation. Regional wall motion and curvature are calculated from these data and are temporally and spatially distributed over 64 chords and 10 deciles, respectively. The maps (represented by a 2D array) are then created for each parameter. Each array element is represented by wall motion and curvature values. Multicolor display allows simultaneous viewing of both parameters. Derivation and depiction of contour maps from one cardiac cycle takes <15 min. We tested this approach in 7 dogs undergoing two-dimensional echocardiographic examination at baseline and during ischemia and found it to correlate well with expert assessment of wall motion.

In conclusion, we have developed a computer-aided approach for the spatial and temporal quantitation of regional endocardial contour and motion using two-dimensional echocardiography. Because this approach does not require outlining the entire endocardial contour, it could be used clinically for assessment and display of regional contraction patterns.

TRANSPUTER MODELLING OF VENTRICULAR DYSRHYTHMIA

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A left ventricular two dimensional model based on a simple finite element approach has been adopted for transputer (a multiprocessor capable of expressing parallelism) simulation. The model's electrical stability is examined as a function of the following parameters: ventricular simulation rate, conduction velocity, mean refractory period (consisting of absolute and relative refractory periods), standard deviation of refractory period and excited state period. A B003 transputer board containing 4 transputer nodes, has been used for processing the model. The speed of the program is enhanced by having the model's cells divided into three transputer nodes, with the fourth node responsible for screen updating. Cells on the screen are represented graphically as points and a resultant electrocardiogram (ECG) of the state of the cells is plotted by dipole summation. The model can display a variety of rhythm disturbances starting with a normal rhythm changing into tachycardia and finally fibrillation before it is returned to its normal state by defibrillation. The influence of sub-groups of cells (i.e. their size and location) with different characteristics to the normal cells necessary to create fibrillation is described. The aim of this paper is to introduce concurrency and implement a computer model which will include the other heart chambers and conduction mechanisms such as Atrioventricular Node, Sino Atrial Node, and His-Purkinje fibres, with no serious compromise on the speed of execution of the program. This style of computer modelling will lay the fundation for future models that need to incorporate complexity for real system simulation.

COMPUTER SIMULATION OF VENTRICULAR EXCITATION USING THE PETRINET METHOD TO ANALYZE THE VENTRICULAR TACHYARRHYTHMIA

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Much work has been done to analyze the mechanism of the ventricular tachyarrhythmia using many experimental models. However, the mechanism of ventricular tachycardia or fibrillation (VTF) induced by electric shock given at the vulnerable period is uncertain. We developed a computer simulation of ventricular excitation using a new myocardial model in order to analyze the mechanism of the cardiac tachyarrhythmia and tested the hypothesis that prolonged refractoriness is the main mechanism of strong energy shock to the induction of VTF. Myocardial model consists of 2116 cell units, formed as a two dimensional model with 46×46 cells. Each cell model takes either of activation phase; excited, refractory, or repolarized. In cardiac tachyarrhythmia, the abnormalities of cardiac excitation propagation are existed in many part of myocardium simultaneously. We used the petrinet method in this model, since it is a modeling method developed to represent the system which has concurrency and parallelism. In the simulation, pacing was started at the middle of one side of the model with the cycle length of 400 ms and the conduction velocity was 2 ms per cell. Electric shock was given at the center of the model with various coupling interval and energy strength. When shock was given at the refractory period of the activation phase the refractory period was set to be prolonged for each cell depending on the distance from the shock site and the energy strength. VTF was observed when certain amount of shock was delivered at certain coupling interval from 175 ms to 210 ms. At VTF episode, the simulation showed activation front moving with distortion, avoiding the area of prolonged refractory period, then marched dividingly, merged, and made vortexes. This result was compatible to the experimental result reported previously. VTF was never induced when prolonged refractoriness mechanism was not indicated.